SOUTHERN DISTRICT OF TEXAS
GALVESTON DIVISION

SARAH PALMQUIST ET AL., *Plaintiffs*, v. THE HAIN CELESTIAL GROUP, INC., *Defendant*.	)	Civil Action No. 3:21-CV-90

PART 1 OF PLAINTIFFS' OMNIBUS RESPONSE TO DEFENDANT'S MOTIONS TO EXCLUDE CERTAIN TESTIMONY OF PLAINTIFFS' EXPERTS

In response to Hain's two consolidated motions to exclude filed August 19, 2022—Hain's Motion to Exclude Expert Testimony on General Causation (ECF 50) and Motion to Exclude Expert Testimony on Specific Causation (ECF 53)—Plaintiffs file two consolidated briefs, although in a format different from Hain's. Rather than separating general causation from specific causation, Plaintiffs found it best to first address generally applicable information and arguments and then arguments specific to each challenged witness. For ease of reading, Plaintiffs have broken up these arguments into two briefs, which are intended to read seriatim.

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INTRODUCTION

Hain's motion for summary judgment and supporting omnibus motions to exclude amount to 100+ pages of building up and then tearing down a strawman. In page after page, Hain tries to undermine any connection between autism spectrum disorder (autism or ASD) and exposure to heavy metals such as those indisputably present in its tainted products. But this case is not and never has been about autism, let alone what may generally or specifically cause it. That is a case of Hain's own creation.

Plaintiffs' case is instead one about brain injury caused by heavy metals poisoning. Plaintiffs' son, Ethan, "went from a vibrant, active, and talkative toddler to suffering from concrete and sustained abnormalities that would be diagnosed as brain damage resulting from confirmed heavy metal toxicity." Compl. ¶ 29 (ECF 6). And Hain is the defendant because its "products have severely and permanently damaged [Ethan]'s brain and neurological function, . . . resulting in profound developmental delay and intellectual impairment." *Id.* ¶¶ 3, 37. Hain has thus briefed a case that does not exist, at least not in this Court.

Hain's strategy of misdirection is perhaps understandable: It has difficult facts to overcome. It is generally accepted and well-established that heavy metals poisoning can damage the developing brain and result in the very disorder that best captures the whole of Ethan's condition: *intellectual disability* (or ID).[1] In fact, that causal connection is so firmly

[1] The only difference between intellectual disability and major neurocognitive disorder, an additional label for Ethan's condition, concerns the baseline and age of onset. *See infra*, pp. 7–9. The diagnosis of major neurocognitive disorder reflects that Ethan's intellectual disability represents a *decline* in functioning after normal early development. *See id.*

established that even the DSM-5 MANUAL—the primary guide for assigning diagnoses to symptoms of mental disorder—which does not usually speak to causes, advises that "[p]ostnatal causes" of intellectual disability "include toxic metabolic syndromes *and intoxications (e.g., lead, mercury)*." AM. PSYCHIATRIC ASS'N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS 39 (5th ed. 2013) (emphasis added).[2]

Meanwhile, Ethan's heavy metals poisoning has been established by direct proof, including medical evaluation and diagnostic testing. And the only plausible source of that poisoning is Hain's concededly tainted products, which made up nearly the entirety of Ethan's early childhood diet.

Rather than confront these difficult facts establishing its liability, Hain picks misdirection, inventing and briefing the case it wishes it were defending. Even if understandable, however, Hain's subterfuge cannot carry the day. Indeed, because Hain carefully avoided the theory Plaintiffs pleaded and proved up, its briefing is largely irrelevant. Having failed to address Plaintiffs' case, Hain has waived any challenge to the opinions of Plaintiffs' experts that Hain's "products have severely and permanently damaged [Ethan]'s brain and neurological function." Compl. ¶ 3.

At all junctures, the collective causation testimony of Plaintiffs' experts is easily admissible because it is both relevant and reliable. The connection between heavy metals poisoning, brain damage, and conditions like Ethan's is not just generally accepted—as shown by the DSM-5 MANUAL, it is gospel. Plaintiffs' toxicology experts gild the lily with

[2] In the medical context, *poisoning* is synonymous with *intoxication*. *See Intoxication*, STEDMAN'S MEDICAL DICTIONARY (2014), https://tinyurl.com/4x2tea32.

their detailed explanations of the biomechanics involved. And the connection between *Ethan's* heavy metals poisoning and *Ethan's* condition was made here by the application of extensive clinical experience in neurodevelopment (and maldevelopment) to the pattern and presentation of Ethan's condition in the context of extensive diagnostic testing. This same evidence reliably implicates Hain's tainted baby food, as it indisputably contained harmful levels of toxic heavy metals and is the *only* plausible source of Ethan's heavy metals poisoning. Here again Plaintiffs' toxicology experts confirm what is already clear, this time with calculations showing just how dangerous Hain's products were.

In this first of two briefs responding to Hain's omnibus motions to exclude, Plaintiffs will cover facts and issues that cut across all its causation witnesses—a recitation of the facts, a discussion of the legal framework for applying Rule 702, and a demonstration of some of the global errors that fatally undermine Hain's motions to exclude.

FACTUAL BACKGROUD

Given Hain's misleading framing of this case, Plaintiffs recount the facts necessary for an accurate assessment of causation. Ethan's present condition represents a devastating decline in neurological function, particularly as to his cognitive abilities. Unsatisfied with a preordained and, at best, uninformative label of Ethan as autistic, Ethan's parents—including his medical-doctor mother—went looking for a cause. They did so not to find someone to sue, but to find the best treatment for Ethan.

The choice by Ethan's parents was backed by science. When a child develops normally through early life and then suddenly, dramatically loses language, motor, and cognitive function, a brain injury with an identifiable cause is usually at work. In the search

for that cause, Ethan underwent significant diagnostic testing, an MRI, multiple EEGs, a lumbar puncture, metabolic testing, and thorough genetic analyses. None could explain his condition.

Because heavy metal toxicity is a known cause of cognitive impairments, Ethan also underwent testing for heavy metals and their effects multiple times beginning in late 2017. That testing showed multiple signs of damaging levels of heavy metals in Ethan's body. Ethan's biomarkers suggested heavy metal poisoning, and the pattern and presentation of his condition as well as the negative findings from the battery of other diagnostic tests confirmed that indication. Ethan's parents finally had a likely medical cause of his condition.

Heavy metals toxicity is an environmental disease, and so, unlike like other medical conditions that can injure the brain, it has an external cause. Finding that cause is a vital part of the treatment for heavy metals toxicity. For years, this piece of the puzzle of Ethan's condition was missing. The puzzle was completed by a bombshell congressional report in 2021 establishing that baby foods—and Hain's in particular—were "tainted with dangerous levels of arsenic, lead, cadmium, and mercury." Given that Ethan consumed Hain's products almost exclusively from the date of his birth through his third year of life, a critical period of brain development, Ethan's parents had key, devastating information on the cause of Ethan's injuries.

I. Ethan's Decline Into Severe Disability

"Ethan is gravely and permanently disabled." Hain Ex. 7, Nelson Report 5. He will never live independently, graduate high school, or be gainfully employed. Hain Ex. 28,

Settles Report 16. Instead, for the rest of his life, Ethan will require around-the-clock care and supervision. *Id.*

Yet Ethan began his life as a neurotypical, and typically developing, child. As described by Hain's expert, Dr. Alexander Kolevzon: "He sat at 6 months, crawled by 10 months, and was walking by 12 months. His first word was spoken before 12 months-old and 2-word phrases occurred by 12-months. He reportedly pointed by 12 months and engaged in imaginary play by 18 months." Hain Ex. 42, Kolevzon Report ¶ 147. The records from Ethan's pediatrician show the same: No concerning behavior was noted during well-check visits every few months after birth and then at 18, 24, and 30 months. Pls' Ex. 2, Dr. Lasics' Records at R.3082-98.

Then, suddenly, around 30 month of age, Ethan's brain gave way, resulting in "a significant regression [] in social, language, and behavior[.]" Hain Ex. 42, Kolevzon Report ¶ 147. In May 2017, six weeks after his normal 30-month well-check, Ethan's parents returned to the same pediatrician for evaluation of his declining fine motor skills. Pls' Ex. 2, Dr. Lasics' Records at R.3071-82. The pediatrician now had significant concerns about Ethan, including that he showed signs of autism. *Id.*

Today, Ethan is "nonverbal" and suffering from "severe to profound" intellectual disability. Hain Ex. 28, Settles Report 7. He has also lost toileting and fine motor skills. *See* Hain Ex. 42, Kolevzon Report ¶ 154; Pls.' Ex. 3, Dr. Koshy's Records at R.1156, Hain Ex. 28, Settles Report 2-3.

Ethan's current condition runs the gamut and reflects his neurological injury. Along with chronic gastrointestinal issues, Ethan suffers from an epileptiform disorder (excessive

and abnormal brain activity), hypotonia (abnormally decreased muscle tone), and deficits in fine motor skills. Hain Ex. 28, Settles Report 3. Ethan's treating physicians have also diagnosed him with heavy metal intoxication based on biomarkers showing high levels of toxic heavy metals in his system and symptoms consistent with heavy metal intoxication. *Id.*; Hain Ex. 14, Nikogosian Report 4; Hain Ex. 36, Megson Dep. 44:13–45:14. Ethan has also been diagnosed with a range of mental disorders, principally including major neurocognitive disorder, intellectual disability, and autism, as well as anxiety and aggression. Hain Ex. 28, Settles Report 3, 16.

II. Ethan's Mental Disorder

As Hain's briefing focuses almost exclusively on Ethan's ASD diagnosis, Plaintiffs offer an accounting of how mental disorders relevant here are typically diagnosed, as well as how Ethan's condition was diagnosed by each of the three clinicians who were the only ones to formally evaluate his mental functioning: Drs. Anson Koshy, Lisa Settles, and Alexander Kolevzon.

A. Diagnosing Intellectual Disability, Neurocognitive Disorder, and Autism Spectrum Disorder.

The DSM-5 MANUAL—home to neurocognitive disorder (or NCD), intellectual disability, and autism—defines a "mental disorder" as "a *syndrome* characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning." DSM-5 MANUAL at 20 (emphasis added). A *disorder* or a *syndrome* are distinct from a *disease*. A disorder or a syndrome describes "a

constellation of signs and symptoms"; while a disease "is an involuntary physiological or biological illness that typically has some underlying cause." Destiny Peterson & Jared W. Keeley, *Syndrome, Disorder, and Disease* 1, *in* THE ENCYCLOPEDIA OF CLINICAL PSYCHOLOGY (Robin L. Cautin & and Scott O. Lilienfeld eds. 2015).[3] As these definitions show, a disease (e.g., cancer, coronary artery disease) is necessarily connected to the physical and tangible. *Id.* at 3. Disorders, on the other hand, "are inherently societal constructs." *Id.* at 3.

True to the nature of mental disorder diagnoses as labels for constellations of symptoms, the DSM-5 MANUAL aims "first and foremost" to be an "official nomenclature" for "clinical practice" that allows clinicians "to communicate the essential characteristics of mental disorders presented by their patients." DSM-5 MANUAL at xli. "Descriptive validity is what the DSM provides. It allows people to describe or classify phenomena in reliable ways." Teneille R. Brown, *From Bibles to Biomarkers: The Future of the DSM and Forensic Psychiatric Diagnosis*, 2015 UTAH L. REV. 743, 763 (2015). As labels for constellations of symptoms, diagnoses under the DSM-5 MANUAL are not made based on blood tests or other biomarkers, but by cataloging a patient's symptoms and comparing them to the diagnostic criteria set for each disorder classification. DSM-5 MANUAL at 21.

1. Intellectual Disability. Intellectual disability "is characterized by deficits in general mental abilities, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience." DSM-5 MANUAL at 31. "The

[3] Non-legal sources that may be difficult to locate are included in the Appendix.

deficits result in impairments of adaptive functioning, such that the individual fails to meet standards of personal independence and social responsibility in one or more aspects of daily life, including communication, social participation, academic or occupational functioning, and personal independence at home or in community settings." *Id.* Impaired communication is a frequent feature of intellectual disability, as are "difficulties in self-management of behavior." *Id.*

Although intellectual disability "is a heterogeneous condition with multiple causes," known "[p]ostnatal causes include . . . toxic . . . intoxications (e.g., lead, mercury)." *Id.* at 39.

2. Major Neurocognitive Disorder. "The NCD category encompasses the group of disorders in which the primary clinical deficit is in cognitive function, and that are acquired rather than developmental." DSM MANUAL 5 at 591. Neurocognitive disorder and intellectual disability have significant overlap as criteria for both focus on cognition, "mental activities associated with thinking, learning, and memory." *Cognition*, STEDMAN'S MEDICAL DICTIONARY (2014 update), https://tinyurl.com/yypvffv9; *compare* DSM MANUAL 5 at 602 (diagnostic criteria for NCD), *with id.* at 33 (diagnostic criteria for intellectual disability).

Only the person's baseline distinguishes neurocognitive disorder from intellectual disability. The DSM-5 MANUAL provides that, while "disorders whose core features are cognitive are included in the NCD category," "[t]he NCDs are those in which impaired cognition has not been present since birth or very early life, and thus represents a decline

from a previously attained level of functioning." DSM-5 MANUAL at 591.[4] And in discussing intellectual disability, the DSM-5 MANUAL further provides that intellectual disability is "distinct from the neurocognitive disorders, which are characterized by a *loss* of cognitive functioning." *Id.* 40 (emphasis added). Thus, "[a] careful clarification of the individual's baseline status . . . help[s] distinguish an NCD from a . . . neurodevelopmental disorder[]." *Id.* at 610.

"[T]he term *neurocognitive disorder* is widely used and often preferred for conditions affecting younger individuals, such as impairment secondary to traumatic brain injury or HIV infection." *Id.* at 591. Indeed, while NCD is distinct from neurodevelopmental disorders, the two "often co-occur[]" because childhood brain injury "may also lead to significant developmental and learning issues." *Id.* at 610–11. Thus, because cognitive decline "in childhood and adolescence may have broad repercussions for social and intellectual development, . . . intellectual disability . . . and/or other neurodevelopmental disorders may also be diagnosed to capture the full diagnostic picture and ensure the provision of a broad range of services." *Id.* at 609.

3. Autism Spectrum Disorder. An autism diagnosis requires the satisfaction of five criteria. "The essential features of autism spectrum disorder are persistent impairment in reciprocal social communication and social interaction (Criterion A), and restricted, repetitive patterns of behavior, interests, or activities (Criterion B). These symptoms are

[4] In turn, major neurocognitive disorder is distinguished from mild based on the significance of the decline from prior functioning and interference with independence in everyday activities. DSM-5 MANUAL at 602–05.

present from early childhood and limit or impair everyday functioning (Criteria C and D)." *Id.* at 53.

But simply meeting those symptom-based criteria are not sufficient for an autism diagnosis. *Id.* That is because the symptoms that mark ASD "would be expected to occur to some extent in all individuals with intellectual disability." Audrey Thurm et al., *State of the Field: Differentiating Intellectual Disability From Autism Spectrum Disorder*, 10 FRONTIERS PSYCH. article 526, at 1 (2019). Thus the evaluator must (at Criterion E) ensure that those symptoms are "not better explained by intellectual disability . . . or global developmental delay," DSM-5 MANUAL at 51.

In patients with intellectual disability, then, "[a] diagnosis of autism spectrum disorder . . . is appropriate when social communication and interaction are *significantly impaired* relative to the developmental level of the individual's nonverbal skills (e.g., fine motor skills, nonverbal problem solving)." *Id.* at 58 (emphasis added).

B. Ethan's mental disorder diagnoses.

Three clinicians—Drs. Anson Koshy, Lisa Settles, and Alexander Kolevzon—have formally evaluated the symptoms that comprise Ethan's condition for a mental disorder diagnosis.[5] Drs. Koshy and Kolevzon concluded that autism best described Ethan's symptoms. Pls.' Ex. 3, Dr. Koshy's Records at R.1213; Hain Ex. 42, Kolevzon Report ¶¶ 153, 160–63. Dr. Settles disagreed. To her, Ethan's autistic traits are "nonspecific," i.e, not meaningful as a standalone disorder, but merely part of his major neurocognitive

[5] *See infra*, pp. 30–31, for a discussion on others' use of the autism label in this case.

disorder, which has a core feature of "severe to profound intellectual disability." Hain Ex. 40, Settles Dep. 87:22–88:14, 153:16–155:5; Hain Ex. 28, Settles Report 7, 16.

1. Dr. Koshy's diagnosis. Following the May 2017 visit where Ethan's unusual behavior was first noted by his pediatrician, Dr. Lasics, she diagnosed him with "[s]uspected autism disorder," and referred him to the "Autism Center"—the Pediatric Center for Autism and Related Conditions. Pls. Ex. 3, Dr. Koshy's Records at R.1144–46. At the Autism Center, the Center's medical director, Dr. Koshy, diagnosed Ethan with autism. *Id.* at R.1213.

According to his records, Dr. Koshy's evaluation of Ethan amounted to, at most, 20 minutes: 70 of the 90 minutes Ethan was seen were consumed by "formal speech language testing by" someone else. *Id.* at R.1162. Ethan returned a week later for 60 minutes, all of which "were spent reviewing the diagnosis, prognosis, testing results, referrals and treatment plan, school based recommendations, and overall coordination or care." *Id.* at R.1217.

Dr. Koshy's diagnosis of Ethan depended solely on the symptom-based diagnostic criteria for autism (Criteria A and B). In his report, Dr. Koshy concludes that Ethan "did meet the diagnostic criteria for ASD," *id.* at R.1211, and then supplies several checked boxes corresponding to only the DSM-5 MANUAL's Criteria A and B. *Compare id.* at R.1212; *with* DSM-5 MANUAL at 50.

Thus, Dr. Koshy diagnosed Ethan with autism without satisfying Criterion E: "These disturbances are not better explained by intellectual disability." DSM-5 MANUAL

at 50.[6] When later asked "what is autism spectrum disorder?," Dr. Koshy responded with only Criteria A–D. *Compare* Hain Ex. 113, Koshy Dep. 18:19–19:4, *with* DSM-5 MANUAL at 50–51. He then acknowledged that he lacked "insight into Ethan's development" when he diagnosed Ethan with autism. Hain Ex. 113, Koshy Dep. 43:19–44:16.

2. Dr. Settles' Diagnosis. Dr. Settles performed a comprehensive developmental evaluation of Ethan, including substantial observation, a review of Ethan's medical records, and multiple formal psychological tests. *See* Settles Report 2. Dr. Settles concluded:

> Ethan's development is so significantly impacted that his intelligence is estimated to be in the severe-to-profound range of intellectual disability. Ethan is no longer using language to communicate. His interactions with the world are focused on sensory seeking behaviors, repetitive behaviors, and problematic behaviors of concern.

Id. at 16. As Dr. Settles further determined, "Ethan's mental/developmental age would place him at an age younger than 2 years, yet his chronological age is 7 years 5 months, which would estimate him to be in the severe-to-profound range of intellectual disability." *Id.* at 2.

Dr. Settles' principal diagnosis for Ethan was "Major Neurocognitive Disorder of severe intensity." *Id.* at 16; *cf.* DSM-5 MANUAL at 605 ("severe" specifier indicates full dependence on others for the tasks of daily living). Dr. Settles explained that, while "severe

[6] Dr. Koshy did not evaluate Ethan's developmental level at all, *see* Pls. Ex. 3, Dr. Koshy's Records at R.1160; Hain Ex. 113, Koshy Dep. 43:19–44:16, yet his records show a then-recent evaluation by the local early childhood intervention provider, who placed Ethan's cognitive level well below his level of social communication, Pls. Ex. 3, Dr. Koshy's Records at R.1153; Pls.' Ex. 6, ECI Records at R.6820.

to profound intellectual disability" is the core of Ethan's condition, her diagnosis of "major neurocognitive disorder" best describes Ethan's "decline in functioning." Settles Dep. 88:1–91:13.[7] While Dr. Settles further "concurred with the diagnosis of autism," *id.* 87:25–88:1, she explained that Ethan's autistic symptoms are "nonspecific"—that is, not meaningful—because they merely reflect his severe to profound intellectual disability, *id.* 106:3–107:2, 154:5–155:5.

3. Dr. Kolevzon's diagnosis. Dr. Kolevzon evaluated Ethan, *see* Hain Ex. 42, Kolevzon Report ¶¶ 143–50, and concluded that "Ethan meets the criteria for ASD based on the records reviewed and my own examination, including social and communication impairment and restricted behaviors," *id.* ¶ 153. Dr. Kolevzon concluded that Ethan's "adaptive functioning" is very low, *see id.* ¶ 144, and Ethan's "cognition" is "severely impaired," *id.* ¶ 150. Nonetheless, Dr. Kolevzon concluded, without elaboration, that Ethan's "social communication is below what would be expected based on his general developmental level despite the comorbid diagnosis of ID." *Id.* ¶ 161. In concluding that autism best describes Ethan's condition, Dr. Kolevzon made no finding that Ethan's "social communication and interaction are *significantly impaired* relative to the developmental

[7] In deposing another of Plaintiffs' experts, Dr. Stephen Nelson, Hain's counsel misrepresented the authorities relied on by Dr. Settles. Hain's counsel represented that, "in reaching [her diagnosis of major NCD, Dr. Settles] was not applying the criteria that had been laid out in the DSM-5." Hain Ex. 37, Nelson Dep. 151:4–9. What Dr. Settles said was that she was not relying *exclusively* on the DSM-5 MANUAL: "It's based partly on the DSM-5, but also just sort of typical practice." Hain Ex. 40, Settles Dep. 89:7–11. Dr. Settles' evaluation of Ethan and her description of NCD ("acquired loss of cognitive abilities," *id.* 90:8–9) fit the DSM-5 MANUAL to a T. *See supra*, pp. 8–9.

level of the individual's nonverbal skills (e.g., fine motor skills, nonverbal problem solving)." DSM-5 MANUAL at 58 (emphasis added).

Dr. Kolevzon further disagreed with Dr. Settles' diagnosis of major neurocognitive disorder. Dr. Kolevzon said that, in his "view, Ethan's condition does not meet the criteria for any of the neurocognitive disorders." Hain. Ex. 42, Kolevzon Report ¶ 163. Dr. Kolevzon, however, never discussed the criteria, although he did find that Ethan's cognition is severely impaired and a decline from previous functioning. *Compare id.* ¶¶ 147, 150, *with* DSM MANUAL 5 at 591 ("The NCDs are those in which impaired cognition has not been present since birth or very early life, and thus represents a decline from a previously attained level of functioning."). The only reason Dr. Kolevzon offered for ruling out neurocognitive disorder was his view that "major neurocognitive disorder is largely synonymous with dementia (DSM-5) and not relevant to Ethan's case." *Id.* ¶ 162. But when "[t]he DSM-5 introduced the term neurocognitive disorder," it "avoided" using "dementia" specifically because its "use is closely associated with geriatric disorders, while *NCD* encompasses acquired cognitive impairment of all causes at all ages." Mary Ganguli, *Can the DSM-5 framework enhance the diagnosis of MCI?*, 81 NEUROLOGY 2045, 2046 (2013); *see supra*, pp. 8–9.

III. Investigating the Cause of Ethan's Condition.

Once the fog of disbelief and shock of Ethan's initial diagnosis lifted, Ethan's parents and his treating physicians began to search for an underlying cause. And with good reason. "The vast majority of people with profound intellectual disability have identifiable organic causes for their delay." Bryan H. King et al., *Intellectual Disability*, *in*

COMPREHENSIVE TEXTBOOK OF PSYCHIATRY 3498, 3499 (Benjamin H. Sadock et al. eds., 10th ed. 2017) (TEXTBOOK OF PSYCHIATRY).[8] In short, an identifiable disease or trauma that damaged the tissues in their brains.

Moreover, while regression in social and communication skills is common with autism, a social-communication regression after a child's second birthday is "atypical." AM. ACAD. PEDIATRICS COUNCIL ON CHILDREN WITH DISABILITIES, SECTION ON DEVELOPMENTAL & BEHAVIORAL PEDIATRICS, *Identification, Evaluation, and Management of Children With Autism Spectrum Disorder*, 145 PEDIATRICS e20193447, at 17 (2020) (AAP GUIDE). Still more unusual is a decline into a *severe* social-communication deficit. As the DSM-5 MANUAL notes, autism symptoms "may be . . . noted later than 24 months if the symptoms are *more subtle*." DSM-5 Manual at 55 (emphasis added). Severe symptoms should be notable much earlier. *See id.*

Regardless of age, a decline beyond autistic traits (like Ethan's) is itself "rare" and "atypical." *Id.*; AAP GUIDE at 17. Ethan's condition stacks rarity upon rarity. According to both the DSM-5 MANUAL and the AAP GUIDE, these rare features "warrant[] more extensive medical evaluation" because they suggest a patient is suffering from an underlying medical condition that is expressing itself outwardly as autistic traits. DSM-5 MANUAL at 55–56; AAP GUIDE at 17; *see also* AM. PSYCH. ASS'N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS: FIFTH EDITION TEXT REVISION 63 (2022)

[8] "Organic cause" is a medical term meaning "nonidiopathic," that is, the result of "anatomic lesions" (damage to tissue). David Benrimoh et al., *Why We Still Use "Organic Causes": Results From a Survey of Psychiatrists and Residents*, 31 J. NEUROPSYCHIATRY & CLINICAL NEUROSCIENCES 57, 57, 62 (2019).

(revision of DSM-5 MANUAL advising that late regression and regression beyond autistic traits is "rare" and associated with "encephalopathic [brain-injury] conditions").

A. Ethan's diagnostic testing

The only additional medical evaluation Dr. Koshy recommended was a "genetics consultation." Pls.' Ex. 3, Dr. Koshy's Records at R.1215. Because of the severity of Ethan's condition, there was reason to expect "detectable genetic defects" if such defects were present. Hain Ex. 43, Geschwind Report ¶ 66. Accordingly, Ethan and his parents underwent comprehensive genetic testing that revealed no concerning genomic variation. Pls.' Ex. 1, Torkamani Decl. ¶¶ 19–20; Pls.' Ex. 1-C (results of 2018 analysis); *cf.* ECF 51 at 24–26 (discussing Ethan's genetic testing). These results have since been reanalyzed twice without finding any concerning genomic variation. Pls.' Ex. 1, Torkamani Decl. ¶ 19.d (discussing 2022 reanalysis); Pls.' Ex. 1-C at R.2865–68 (results of 2021 reanalysis).

Ethan's parents also sought neurological examination for Ethan. An MRI revealed no abnormalities, further excluding many organic causes of Ethan's condition. Ethan underwent four EEG studies. While the first (taken at 3 years and 3 months) was normal, the remaining three (the first of which was taken around a year later) were abnormal, marking Ethan's rapid decline. Hain Ex. 7, Nelson Report, at 3. None, however, could explain Ethan's condition. Ethan's treating neurologists also ordered a lumbar puncture to examine Ethan's cerebrospinal fluid for signs of an autoimmune disorder. None was found. *See* Pls.' Ex. 15.

Ethan also underwent metabolic and heavy metals testing, as recommended by his treating physician. The AAP GUIDE advises that where "atypical regressions (later than 2

years of age, motor regression, or multiple regressions)" like Ethan's are seen, the patient's autistic traits may be caused by "metabolic or mitochondrial disorders." AAP GUIDE at 17. And, as one of the two review articles relied on by the AAP GUIDE for this proposition explains, these metabolic and mitochondrial dysfunctions may be triggered by heavy metals intoxication. *See* Nassim Zecavati & Sarah J. Spence, *Neurometabolic Disorders and Dysfunction in Autism Spectrum Disorders*, 9 CURRENT NEUROLOGY & NEUROSCIENCE REPORTS 129, 131–33 (2009) (cited by AAP GUIDE at 17 & n.168).[9]

Ethan's metabolic panel was uninformative, but his heavy metals testing suggested heavy metals intoxication—a known cause of intellectual disability. To that end, Ethan's providers obtained a hair test and three urine porphyrin tests, which demonstrated toxic levels of arsenic, lead, and mercury as a result of consuming Hain's products. *See* Hain Exs. 45 (Nov. 2017 Hair Test), 52 (Dec. 2017 Porphyrin Test), 53 (Sept. 2018 Porphyrin Test), 54 (Oct. 2019 Porphyrin Test).

Without any other plausible explanation, Ethan's treating physicians eventually diagnosed him with heavy metals intoxication based on biomarkers—including those flagged by his heavy-metals testing—showing toxic levels of heavy metals in his body and symptoms consistent with heavy metals intoxication. Hain Ex. 28, Settles Report 3; Hain Ex. 14, Nikogosian Report 4; Hain Ex. 36, Megson Dep. 44:13–45:14. "Heavy metal

[9] *See also Metabolic Encephalopathy*, STEDMAN'S MEDICAL DICTIONARY, https://tinyurl.com/bde92u57 ("[S]econdary metabolic encephalopathy results when brain metabolism is disturbed by extracerebral disorders causing intoxication, electrolyte imbalances, or nutritional deficiencies, *e.g.*, hepatic or renal disease *or exogenous poisons*.") (emphasis added).

poisoning [or intoxication] refers to when excessive exposure to a heavy metal affects the normal function of the body." Nat'l Insts. Health, Genetic & Rare Disease Info. Ctr., HEAVY METALS POISONING, https://perma.cc/8337-Y545 (Nov. 8, 2021).

B. Congressional revelations

As "most diseases related to hazardous exposures in . . . children manifest as common medical problems," it is easy to "miss opportunities to make correct diagnoses or prevent disease." U.S. Dep't for Health & Hum. Servs., Agency for Toxic Substances & Disease, *Taking a Pediatric Exposure History* 25 (June 3, 2011), https://perma.cc/66YW-G2ST. As explained, Ethan's parents did not miss the opportunity to identify the immediate cause of Ethan's neurologic damage: heavy metals toxicity, a known cause of intellectual disability. But the source of Ethan's heavy metals poisoning remained unclear, as Ethan had no identifiable source of heavy metals exposure.

Then, in February and September 2021, Congress revealed the source: Hain's baby food products, which Ethan had consumed heavily and almost exclusively from the date of his birth through his third year of life. Pls.' Ex. 4, July 28, 2022 Aff. of Sarah Palmquist at 2 (Ethan "was fed Earth's Best products almost exclusively from his date of birth, September 27, 2014, through the first three years of his life"). The first report—succinctly titled "Baby Foods Are Tainted with Dangerous Levels of Arsenic, Lead, Cadmium, and Mercury"—revealed that Hain exceeded the limits for each of the four metals it tested:

- Hain (Earth's Best Organic) sold finished baby food products containing as much as 129 ppb inorganic arsenic. Hain typically only tested its ingredients, not finished products. Documents show that Hain used ingredients testing as high as 309 ppb arsenic.

- Hain (Earth's Best Organic) used ingredients containing as much as 352 ppb lead. Hain used many ingredients with high lead content, including 88 that tested over 20 ppb lead and six that tested over 200 ppb lead.

- Hain (Earth's Best Organic) used 102 ingredients in its baby food that tested over 20 ppb cadmium. Some tested much higher, up to 260 ppb cadmium.

- Beech-Nut and Hain (Earth's Best Organic) do not even test for mercury in baby food.

Staff Report, Subcomm. On Economic and Consumer Policy of the Comm. On Oversight and Reform, 117 Cong. 3–4 (Feb. 4, 2021), https://perma.cc/9YTR-SH7M; *see* Pls.' Ex. 5, Fajardo Dep. 205:19–210:11.

The second congressional report—"New Disclosures Show Dangerous Levels of Toxic Heavy Metals in Even More Baby Foods"—revealed that "an internal assessment conducted by Hain, the maker of Earth's Best Organic, [ ] found that the company's estimates *underestimate toxic heavy metal levels 100% of the time*." Staff Report, Subcomm. on Economic and Consumer Policy of the Comm. on Oversight and Reform, 117 Cong. 16 (Sept. 29, 2021), https://perma.cc/8PZ4-7C4Q (emphasis added).

With the likely (indeed, the only plausible) source of Ethan's heavy metals toxicity and resulting disability identified, Plaintiffs brought this suit, seeking damages for the destruction of their child's life. Several experts have worked on Plaintiffs' behalf to

confirm the link between Hain's products and Ethan's injury. Many of those experts are now the target of Hain's motions to exclude.

APPLYING FEDERAL RULE OF EVIDENCE 702

Under Rule 702, "trial judges are gatekeepers, not armed guards." 29 CHARLES A. WRIGHT ET AL., FEDERAL PRACTICE AND PROCEDURE § 6268.2 (2d ed., updated June 9, 2022) (WRIGHT & MILLER). Rule 702 has the modest aim of keeping "junk science" from reaching the jury box. *Skidmore v. Precision Printing & Pkg., Inc.*, 188 F.3d 606, 618 (5th Cir. 1999); *see* ECF 51, 61 (Plaintiffs' motions to exclude certain testimony of Hain's experts). Avoiding junk science is not a high bar to clear, and thus "the rejection of expert testimony is the exception rather than the rule." *Puga v. RCX Sols., Inc.*, 922 F.3d 285, 294 (5th Cir. 2019) (quoting FED. R. EVID. 702 advisory committee notes (2000)).[10]

The gatekeeping role for the Court under Rule 702 reflects (1) what "one trained in the law could be expected to do," *id.* § 6262, (2) "the liberal thrust of the Federal Rules and their general approach of relaxing the traditional barriers to opinion testimony," *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 588 (1993) (quotation marks omitted), and (3) what the Constitution demands:

[10] The most illustrative examples of the low bar erected by Rule 702 are found in this Circuit's criminal cases. Even when a defendant's freedom is at stake, there is a "category of cases in which the evidence was shaky but admissible, and the traditional tools of attacking the evidence were the proper means of attack." *United States v. Perry*, 35 F.4th 293, 330 (5th Cir. 2022) (citing examples). In *Perry*, for instance, the challenged expert conceded "that some photographs of her analysis were lost, that there was a point during the examination of the evidence when her lab was unaccredited, and that ballistics was not a science with a mathematical degree of confidence." *Id.* These shortcomings, the court concluded, were for the jury to consider. *Id.*

> [A] court proceeding, such as a trial, is not simply a search for dispassionate truth. The law must be fair. . . . One important procedural safeguard, guaranteed by our Constitution's Seventh Amendment, is the right to a trial by jury. . . . Any effort to bring better science into the courtroom must respect the jury's constitutionally specified role—even if doing so means that, from a scientific perspective, an incorrect result is sometimes produced.

Stephen Breyer, *Introduction* to REFERENCE MANUAL ON SCIENTIFIC EVIDENCE: THIRD EDITION 1, 4 (Nat'l Rsch. Council et al. eds., 2011) (REFERENCE MANUAL). In sum, "the trial court's role as gatekeeper is not intended to serve as a replacement for the adversary system." *United States v. 14.38 Acres*, 80 F.3d 1074, 1078 (5th Cir. 1996).

In turn, the ken of the Court, the liberal thrust of the rules, and the primacy of the jury have several practical implications for the application of Rule 702. Beginning at the end, the primacy of the jury means that "[t]he court must . . . not conflate 'admissible' with 'sufficient.'" *Huss v. Gayden*, 571 F.3d 442, 460 (5th Cir. 2009). Likewise, it is not for the Court to resolve disputes over facts underlying expert testimony. Rather, the "fact-finder is entitled to hear [an expert's] testimony and decide whether . . . the predicate facts on which [the expert] relied are accurate." *Pipitone v. Biomatrix, Inc.*, 288 F.3d 239, 250 (5th Cir. 2002).

The liberal thrust of the Rules of Evidence builds on the primacy of the jury. Because "[t]he Rules were designed to depend primarily upon lawyer-adversaries and sensible triers of fact to evaluate conflicts," *Daubert*, 509 U.S. at 589 (quotation marks omitted), a party need not satisfy the scientific community to get to a jury. The law does not seek or require scientific certainty, but, more humbly, merely "decisions that fall within the boundaries of scientifically sound knowledge." Breyer, *supra*, REFERENCE MANUAL 4.

Thus, "the absence of a scientific consensus on a given theory does not affect the admissibility of an expert's opinion." *Peteet v. Dow Chem. Co.*, 868 F.2d 1428, 1433 (5th Cir. 1989). Instead, even for "shaky" expert evidence, "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attack[]." *Puga*, 922 F.3d at 294 (quoting *Daubert*, 509 U.S. at 596) (quotation marks omitted).

Similarly, given his or her core skills, "[t]he judge is not asked to decide if the expert's opinions are, in fact, scientifically or technically correct since this is more than one trained in the law could be expected to do." 29 WRIGHT & MILLER § 6262; *see also, e.g.*, *Prantil v. Arkema France S.A.*, 2022 WL 1570022, at *21 (S.D. Tex. May 18, 2022) (Ellison, J.) (distinguishing "the accuracy of [the expert's] conclusions" from "the reliability of his approach"). Instead, "the judge should only exclude the evidence if the flaw is large enough that the expert lacks 'good grounds' for his or her conclusion." *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 355 (5th Cir. 2007) (quoting *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 745 (3d Cir. 1994)).

ARGUMENT

It is universally recognized that heavy metals poisoning causes brain damage, especially in children. And chronic exposure to even low levels of lead, arsenic, or mercury consumption is damaging to the developing brain. Following the revelations in the congressional reports, then, there should be no dispute that Hain's baby food, which Ethan consumed exclusively in early life, contained harmful levels of heavy metals. *See* ECF 50 at 1–2 (Hain conceding that its products contained heavy metals); ECF 53 at 11 (same).

All experts here agree that Ethan's condition is the result, most immediately, of damage to his brain. Hain's expert, Dr. Daniel Geschwind, posits that the "neurobiological" mechanism behind Ethan's condition is an alteration in "the number and identity of neurons in the brain, changing the brain structures and their connectivity patterns[,]" which "eventually impacts higher cortical functions." Geschwind Report ¶ 85. And as one other example, Plaintiffs' expert, Dr. Settles, concludes that Ethan's condition is "related to some type of brain injury or brain damage." Settles Dep. 61:25–66.

Where the parties part ways is at the cause of that damage. Plaintiffs submit that the damage is "secondary to heavy metal toxicity, secondary to Earth's Best baby food consumption." Nelson Dep. 141:22–24; *accord* Compl. ¶¶ 3, 29, 37. Hain insists the cause is some variation in Ethan's genes. *See* Answer ¶ 77 (ECF 11) (asserting that some preexisting genetic abnormality caused Ethan's condition); *cf.* ECF 51–52 (challenging Hain's evidence of a preexisting condition).

At worst, this "dispute . . . as to whether [Ethan's] disability resulted from injury" or a preexisting condition "is a classic 'battle of the experts.'" *Cox v. Provident Life & Accident Ins. Co.*, 878 F.3d 504, 507 (5th Cir. 2017). "This battle should be fought with the conventional weapons of cross-examination and competing testimony—not the nuclear option of exclusion." *Williams v. Manitowoc Cranes, L.L.C.*, 898 F.3d 607, 625 (5th Cir. 2018). For a jury to hear an expert's opinion, the party offering the testimony need not satisfy the Court that the opinion is correct, but must merely show by a preponderance of the evidence that the opinion is relevant and reliable. Reliability, in turn, is a matter simply of good grounds—sufficient facts and a rational explanation for how those facts lead to

conclusion. Collectively, the causation testimony of Plaintiffs' experts easily clears that bar.

But before turning to Plaintiffs' experts, it is necessary to clear the underbrush of Hain's multiple, global errors. For one, Hain's entire briefing on causation rests on the false premise that Plaintiffs' case turns on what caused Ethan's autism diagnosis. In fact, Plaintiffs' case turns on what caused Ethan's heavy metals poisoning that injured his brain and left him severely disabled in ways far beyond his loss of social functioning. *See* **Part I**. Plaintiffs' theory has been open and obvious since the case began, but Hain has chosen to ignore it. As a result, Hain's briefing is irrelevant, and it has forfeited any Rule 702 challenge to the opinions of Plaintiffs' experts on the cause of Ethan's severe disability.

Asking the right question leads to an easy answer on general causation: It is generally and widely accepted that heavy metals poisoning can do severe neurologic damage to the developing brain of a child, which expresses in developmental deficits, including in language and speech, motor skills, behavior, memory, learning, and other neurological functions. *See* **Part II**. This fact is recognized by expert organizations, government agencies, and medical textbooks. It is not debatable.

Because general causation is beyond dispute, and for other reasons, it is unnecessary to conduct a Bradford-Hill analysis or other analysis of literature. *See* **Part III**. A Bradford-Hill analysis is never required by Rule 702. Nor must an expert rely on epidemiological literature where no contrary literature is cited. Still, Plaintiffs' experts rely on significant literature establishing that heavy metals poisoning can damage the brain and thus result in intellectual disability.

In the next brief, Plaintiffs will show additional global errors by Hain and confirm the reliability of their witnesses' testimony.

I. Ethan's Autism Diagnosis Is a Mere Distraction and Not the Lens Through Which the Court Must Evaluate the Testimony of Plaintiffs' Experts.

It is well recognized that "[e]vidence of mental disease . . . can easily mislead." *Clark v. Arizona*, 548 U.S. 735, 775 (2006); *accord, e.g.*, DANIEL W. SHUMAN, PSYCHIATRIC AND PSYCHOLOGICAL EVIDENCE § 2:11 (3d ed. 2021) (warning that the "use of psychiatric diagnostic categories may confound rather than clarify"), https://tinyurl.com/bd4u63b8. The authors of the DSM-5 MANUAL concur. They understood that their categories would, as here, end up in a courtroom for one reason or another. And so they warned of the "risk that diagnostic information will be misused or misunderstood," a danger that arises "because of the imperfect fit between the questions of ultimate concern to the law and the information contained in a clinical diagnosis." *Id.*

Where others see danger, Hain sees opportunity. Hain has devoted its whole briefing to discussing the cause of Ethan's autism diagnosis to "confound rather than clarify," SHUMAN, *supra*, § 2:11, to distract from the conclusive evidence establishing its liability. But Hain's want-to cannot change the case that Plaintiffs pleaded and will prove into the case that Hain *wished* Plaintiffs had pleaded and would try to prove.

The "particular disease" for which Plaintiffs will establish a "causal link," ECF 50 at 31 (quotation marks omitted), is not autism—which is not a disease at all, or even an informative diagnosis here. That disease instead is "heavy metal toxicity," Compl. ¶ 29, an environmental disease with a signature cause that was indisputably present here—heavy

metal exposure. Heavy metal toxicity is a well-established trigger of the cognitive deficits that characterize intellectual disability, Ethan's most informative and reliable diagnosis, and a disorder that is often mistaken for autism.

Far from establishing that the Court should exclude Plaintiffs' causation experts, by attacking the strawman of autism, Hain has forfeited any challenge to the reliability of their opinions that Hain's tainted products poisoned Ethan and injured his brain.

A. Hain's focus on autism does not address Plaintiffs' theory of causation.

Hain seizes on a single diagnosis among the many given to Ethan and tries to turn Plaintiffs' case from one about Hain's role in Ethan's "heavy metal toxicity," which "damaged" Ethan's "brain and neurological function, . . . resulting in profound developmental delay and intellectual impairment," Compl. ¶¶ 3, 29, 37, into a debate over what causes "autism"—a word never mentioned in Plaintiffs' Complaint.

Indeed, Hain built its entire defense onto that manufactured edifice. A sampling:

- "Plaintiffs allege that their minor child EP developed autism as a result of eating Earth's Best Baby Food manufactured by Defendant The Hain Celestial Group, Inc." ECF 54 at 1.
- "Plaintiffs' claims rest entirely on the speculative and unproven hypothesis that exposure to trace levels of naturally occurring heavy metals—which are ubiquitous in the environment and food supply—causes autism." *Id.*
- "Plaintiffs have not offered reliable or sufficient evidence to establish that heavy metals in baby foods are capable of causing autism" *Id.* at 7.
- "This motion focuses on . . . whether ingestion of heavy metals present in food, including baby food, can generally cause children to develop autism." ECF 50 at 1.
- "Plaintiffs must separately prove specific causation: that levels of heavy metals present in the baby food that EP ate actually caused his ASD." ECF 53 at 1.

- "Using heavy metal tests to diagnose a cause of autism has no scientific basis." *Id.* at 12 (emphasis omitted; capitalization altered).

As shown, however, Plaintiffs do *not* allege that Ethan "developed autism as a result of eating Earth's Best Baby Food." ECF 54 at 1. Nor do Plaintiffs "rest" at all "on the . . . hypothesis that exposure to . . . heavy metals . . . causes autism." *Id.* For that reason, Plaintiffs have no need to prove "whether ingestion of heavy metals . . . can generally cause children to develop autism." ECF 50 at 1. Or "that levels of heavy metals present in the baby food that [Ethan] ate actually caused his ASD." ECF 53 at 1. Plaintiffs do not need to prove a theory that Hain *wishes* Plaintiffs had pursued.

Hain ignored so many signs that Plaintiffs were not pursuing the theory that heavy metals cause autism that one can only conclude Hain has strategically chosen to be obtuse, hoping Plaintiffs—and ultimately this Court—would agree to this mischaracterization of their case. But none of the reports prepared by Plaintiffs' experts focused on a link between heavy metals and autism. Dr. Aschner mentions autism only in passing during his discussion of the diverse neurotoxic effects of heavy metals. Hain Ex. 1, Aschner Report at 19, 22. As Hain notes, "Dr. Parran's report . . . nowhere . . . mentions autism or ASD." ECF 50 at 28. Likewise, Hain observes that "Dr. Settles . . . and Dr. Nelson do not offer any opinions in their reports that exposure to heavy metals in baby food—or exposure to heavy metals generally—can be a cause of autism." *Id.* at 26.

Ethan's treating physicians, while not retained to necessarily support Plaintiffs' theory, offered similar warnings. Dr. Armen Nikogosian, for instance, was careful to explain Ethan's condition not as autism, but as "complex medical issues," including

"developmental delay," "which in turn are driving his *clinical expression* of severe Autism Spectrum Disorder." Hain Ex. 14, Nikogosian Report at 4 (emphasis added).[11] Dr. Mary Megson, meanwhile, explained that she did not know if Hain's products caused autism. ECF 50 at 28.

Although Plaintiffs' experts discuss autism extensively in their depositions, the reason is straightforward: they were responding to Hain's obtuse questioning. *Cf.* ECF 50 at 26 (complaining about witnesses' "unsupported nebulous musings" in response to Hain's questions about autism). And even there, unmistakable signs warned Hain that it was barreling down the wrong path. Dr. Damani Parran explained that linking heavy metals to an autism diagnosis "wasn't the focus of this work." Hain Ex. 39, Parran Dep. 133:2–14. Dr. Stephen Nelson explained that severity of Ethan's condition was like nothing he had seen before. *See* Hain Ex. 37, Nelson Dep. 112:25–113:8. And Dr. Settles, an expert on neurodevelopmental disorders, tried repeatedly to explain to Hain's counsel that Ethan's autism diagnosis was meaningless:

- "So one of my opinions is that his – his specific expression of autism symptoms is atypical, and that his issues stem from the major neurocognitive issues that he has or the intellectual disability, and that would be related to some type of brain injury or brain damage." Hain Ex. 40, Settles Dep. 64:25–66:5.
- "[M]y opinion is that the issues that are facing Ethan and the things that are of the biggest detriment to him right now are actually more than neurocognitive and intellectual disability, and I think the more severe profound you get on the scale of intellectual disability, that is . . . going to start to show signs and symptoms of

[11] "Global developmental delay, as its name implies, is diagnosed when an individual fails to meet expected developmental milestones in several areas of intellectual functioning." DSM-5 MANUAL at 31.

autism, and I think he has enough of those in order to also attain a diagnosis of autism." *Id.* 106:4–13.

- "I don't think that talking about the issues in this case and looking at heavy metal and autism exclusively, I don't think that really applies to what's going on with Ethan." *Id.* 106:23–107:2.

- "So you can have an intellectual disability without having autism, and as your symptoms of intellectual disability, as you go down in functional capacity, so as you get to the severe and profound level, oftentimes you start to see more symptoms of autism present." *Id.* 154:5–10.

- "So that is an important distinction to make, where are his deficits coming from, and I think that's important in this case because I don't – I don't think that his autism is the major component that needs to be focused on." *Id.* 154:15–19.

Hain ignored these warnings and stubbornly pressed on. Whatever the reason for Hain's dogged focus on autism, it defends a case that Plaintiffs did not plead and need not prove.

B. Ethan's autism diagnosis is uninformative and irrelevant.

On top of ignoring the case Plaintiffs are pursuing, Hain either misunderstands or mischaracterizes the nature of Ethan's autism diagnosis. Ethan's autism diagnosis is meaningless against the backdrop of his entire condition, which as Dr. Settles explained, is best described as acquired severe to profound intellectual disability, or major neurocognitive disorder, a condition that subsumes Ethan's autistic traits. *See also supra*, pp. 12–13. In Hain's more than 100 pages of briefing (ECF 50, 53, 54), they fail to mention Ethan's severe to profound intellectual disability *a single time*, let alone discuss its cause. Hain's silence is likely strategic, as heavy metals toxicity is a known cause of intellectual disability. *See infra*, Part III. Whistling past the graveyard, however, is not an effective defense.

To distract from how little Ethan's autism diagnosis means against the background of his entire condition, Hain treats autism as a *disease* from which Ethan is suffering. *See, e.g.*, ECF 50 at 33 (discussing "the vulnerability period for developing the disease" of autism); *see also* ECF 53 at 14 (suggesting "autism itself can *be a cause* of elevated porphyrin levels") (emphasis added). But autism is not a disease. *See supra*, pp. 6–7 (discussing diseases vs. disorders). It is merely a *label* for someone with abnormally low social communication and social interaction functioning accompanied by repetitive behaviors or interests. DSM-5 MANUAL 50; *accord* ECF 50 at 3.[12] And autism is a particularly slippery label here, where several diagnoses are at play, as "any neurodevelopmental or adult psychiatric condition is now suspected to have autistic traits." Laurent Mottron, Commentary, *A radical change in our autism research strategy is needed: Back to prototypes*, 14 AUTISM RESEARCH 2213, 2215 (2021).

Ultimately, Ethan's autism diagnosis is insignificant to this case because, even as a label, it is uninformative. Hain reveals the lack of information contained in Ethan's autism diagnosis, and how that diagnosis can be used to mislead, when it claims that "Plaintiffs' experts all agree that EP has been properly diagnosed with autism." ECF 54 at 2; *accord* ECF 50 at 13 & n.36. Here is what Hain sees as a useful diagnosis of autism:

- Dr. Michael Aschner, a toxicologist, is "not a physician" and thus does not "question" the diagnoses of others. Hain Ex. 35, Aschner Dep. 68:20–69:15.
- Dr. Ali Torkamani is a biomedical scientist, "not a clinician," did not "have a strong opinion one way or another" about whether Ethan "has ASD," and described

[12] For this reason, Hain's distinction between those with autistic traits and those who have been given the autism label, ECF 50 at 37–39, to the extent it remains relevant at all, is misguided, *see* ECF 51 at 13–14.

Ethan's autistic traits as "a typical sort of autism case" based on "a friend whose child is autistic." Hain Ex. 41, Torkamani Dep. 23:15–24:21; 41:2–42:7.

- Dr. Settles made clear that Ethan's autistic traits are "nonspecific," i.e., meaningless. Hain Ex. 40, Settles Dep. 154:5–155:1.

- Dr. Nelson, who defers to Dr. Settles on mental disorder diagnoses, used the term "severe regressive autism" to *distinguish* Ethan's *condition* from his autism diagnosis—the focus of Hain's questioning. Hain Ex. 37, Nelson Dep. 123:11–126:7, 150:13–151:3 ("[N]ot only does he have autism, but he also has severe to profound intellectual disability with some preservation of social skills."); *accord* Hain Ex. 7, Nelson Report 5 ("[T]here are no identified alternative causes for Ethan's *severe Major Neurocognitive Disorder* other than heavy metal toxicity during the first two years of his life.") (emphasis added).

- Dr. Megson, who concurred with Ethan's initial autism diagnosis based on only a videoconference with his mother. Hain Ex. 4, Megson Report 3.

As these statements show, Ethan's autism diagnosis is no more than a rough shorthand for a small subset of his symptoms (and hardly a substitute for his entire condition).[13] Focusing on Ethan's autism diagnosis lets the tail wag the dog.

The context of Ethan's formal diagnoses confirms that his autism diagnosis is meaningless against the backdrop of his entire condition. With conditions like Ethan's, involving severe to profound intellectual disability, "clinicians are presented with the difficult task of determining when observed social deficits are attributable to an individual's ID, and when an additional diagnosis of ASD is warranted." Thurm et al.,

[13] To the extent Hain's myopic focus on autism shows that Hain believes Ethan's condition is no more than autism—*cf., e.g.*, Pls.' Ex. 14, Wilfong Report 2 (Hain's expert opining that "Ethan's epileptiform discharges are part of his ASD"); Hain Ex. 43, Geschwind Report ¶ 6 (opining that Ethan's "symptoms can all be explained based on him having an ASD diagnosis")—such a belief would be badly mistaken. As Hain rightly concedes, an autism diagnosis describes no more than "persistent deficits in social communication and interaction and restricted, repetitive patterns of behavior, interests, or activities." ECF 50 at 3.

supra, at 3. This difficulty exists not just because deficits in social communication and interaction (Criterion A) "would be expected to occur to some extent in all individuals with intellectual disability." *Id.* at 1. It also exists because repetitive behaviors (autism Criterion B), like Ethan's hand flapping, "are also seen in other conditions, particularly severe intellectual disability," which makes them "[a] common source of [autism] diagnostic error." Fred R. Volkmar et al., *Autism Spectrum Disorder and Social Communication Disorder*, *in* TEXTBOOK OF PSYCHIATRY 3571, 3582–83.

In fact, it is so hard to tease autism out of severe to profound intellectual disability that researchers have begun to exclude from autism studies subjects who share these diagnoses. *See* Thurm et al., *supra*, at 4. "The scientific validity of . . . research findings is influenced by the reliability of the diagnosis . . . under study." Michael D. Green et al., *Reference Guide on Epidemiology*, in REFERENCE MANUAL 549, 589. These researchers understand what Hain pretends not to: An autism diagnosis in the presence of severe to profound intellectual disability is inherently unreliable as a standalone condition.

So too here. In a formal diagnostic setting, to distinguish autism from intellectual disability, "[t]he clinician *must* consider the behaviors expected at a given developmental level." Thurm et al., *supra*, at 4 (emphasis added). Only "when social communication and interaction are *significantly impaired* relative to the developmental level of the individual's nonverbal skills," is an autism diagnosis meaningful. DSM-5 MANUAL 58 (emphasis added).

While Dr. Settles explained this requirement repeatedly, *see supra* pp. 28–29, the other two clinicians to formally evaluate Ethan ignored it. Dr. Koshy agreed that

Ethan presented with "a classic case of severe autism," but he did *not* "confirm[] that [Ethan] met *all* the DSM-5 criteria" (as Hain incorrectly claims), but merely that Ethan's traits met the symptom criteria. ECF 50 at 13 (emphasis added) (citing to testimony in which Dr. Koshy confirms only that Ethan met two of the five criteria for autism).[14] But "it is not sufficient to simply check off the symptoms in the diagnostic criteria to make a mental disorder diagnosis." DSM-5 MANUAL at 19. As Dr. Koshy even conceded, he lacked "insight into Ethan's development" (Criterion E). Koshy Dep. 52:21-25; *see supra*, pp. 11–12. Dr. Kolevzon similarly found no significant impairment in Ethan's social communication and interaction compared to his "severely impaired" "cognition." Hain Ex. 42, Kolevzon Report ¶ 150; *see supra*, pp. 13–14.

In the end, not one piece of evidence here supports treating Ethan's autism diagnosis as anything but a feature of his acquired severe to profound intellectual disability. Thus, Plaintiffs need not establish a cause of Ethan's autism diagnosis beyond the cause of his intellectual disability. *See Burton v. Wyeth-Ayerst Lab'ys*, 513 F. Supp. 2d 719, 724 (N.D. Tex. 2007) ("The issue presented by Wyeth seeks to have the court carve out a distinction between these two diseases essentially as a matter of law. Based on the evidence before the court, such a distinction cannot be drawn."). At worst, even should the Court conclude that there is some basis for treating Ethan's autism diagnosis as distinct from his intellectual disability, the question of whether that diagnosis does, in fact, represent a distinct condition

[14] Dr. Koshy was mistaken even as to his characterization of Ethan's autism diagnosis. Regression into severe autism (to say nothing of decline into accompanying severe to profound intellectual disability) after age two is "unusual" and "atypical," not *classic*, and calls for additional medical evaluation to look for a cause. *See supra*, pp. 15–16.

needing its own cause is "grist for the jury." *Carroll v. Morgan*, 17 F.3d 787, 790 (5th Cir. 1994); *see Skidmore v. Precision Printing & Pkg., Inc.*, 188 F.3d 606, 614 (5th Cir. 1999) (concluding that sufficient supporting symptoms and a formal diagnosis "is sufficient evidence" of the existence of a mental disorder "if the jury chose to credit the testimony").[15]

At all events, Hain's autism-focused briefing is no hurdle to Plaintiffs presenting their claims to a jury.

II. Plaintiffs' General Causation Theory Is Widely Accepted in the Scientific and Medical Communities.

Hain's briefing focuses exclusively on whether Plaintiffs' experts have satisfactorily established that heavy metals can cause autism. Thus, Hain has *not* challenged the causation theory of Plaintiffs and their experts—that heavy metals exposure can cause damaging heavy metals toxicity and resultant intellectual impairment. The deadline for pretrial motions has passed, and "arguments raised for the first time in a reply[] are waived." *Branch v. CEMEX, Inc.*, 2012 WL 2357280, at *9 (S.D. Tex. June 20, 2012) (Rosenthal, J.), *aff'd*, 517 F. App'x 276 (5th Cir. 2013). So Hain's opportunity to challenge Plaintiffs' experts on that ground has elapsed.

[15] *Cf. Ake v. Oklahoma*, 470 U.S. 68, 81 (1985) ("[P]sychiatrists disagree widely and frequently . . . on the appropriate diagnosis to be attached to given behavior and symptoms."); DSM-5 MANUAL's 6, 21 (explaining that "the boundaries between disorders are more porous than originally perceived" and thus "[d]iagnostic criteria are offered as guidelines for making diagnoses, and their use should be informed by clinical judgment"); *id.* at 22 (observing that while "[t]he general convention in DSM-5 is to allow multiple diagnoses to be assigned for those presentations that meet criteria for more than one DSM-5 disorder," the evaluator nonetheless must use clinical judgment to determine the "principal diagnosis," i.e., "the main focus of attention," which is an "often difficult (and somewhat arbitrary)" distinction to make).

That said, solely for the Court's benefit, Plaintiffs will show that their causation theory is widely accepted in the scientific and medical communities and thus that their expert's causation opinions are presumptively reliable. *See Cano v. Everest Mins. Corp.*, 362 F. Supp. 2d 814, 824 (W.D. Tex. 2005) (holding that general acceptance of idea that radiation at any level can cause cancer satisfied general causation); *see also McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1239 (11th Cir. 2005) ("The court need not undertake an extensive *Daubert* analysis on the general toxicity question when the medical community recognizes that the agent causes the type of harm a plaintiff alleges."). The connection between heavy metals toxicity and neurologic damage, including damage resulting in intellectual disability, is recognized and accepted by medical organizations, federal and state government health agencies, and medical educators. There are few connections in the study of environmental disease more widely accepted. Nothing in Hain's briefing suggests otherwise.

A. Authoritative sources universally recognize that heavy metals toxicity causes brain injury that results in intellectual disability.

As the Supreme Court explained in *Daubert*, "[w]idespread acceptance can be an important factor in ruling particular evidence admissible." 509 U.S. at 594. This is especially true in fields like medicine and science, "which have been found to have a vigorous tradition of testing." 4 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY § 32:10 (2021 ed.),

https://tinyurl.com/yc8ekpff.[16] Hain acknowledges the value of general acceptance in establishing reliability, repeatedly looking to what "recognized medical, scientific, or research bod[ies]" recognize as causes of conditions. ECF 50 at 7–8; *see also id.* at 19.

One authority that Hain asks the Court to look to is the American Psychiatric Association. *Id.* at 8. The American Psychiatric Association, which defines intellectual disability, recognizes "exposure to toxins such as lead or mercury" as one of the "causes of intellectual disability." Am. Psych. Ass'n, *Causes*, WHAT IS INTELLECTUAL DISABILITY? (Aug. 2021), https://perma.cc/UB3N-VJCA. The DSM-5 MANUAL, which followed years of study and the input of thousands of doctors and scientists, repeats this known causal pathway. DSM-5 MANUAL at 39 (advising that "[p]ostnatal causes" of intellectual disability "include . . . intoxications (e.g., lead, mercury)").

The nation's leading psychiatric organization is hardly out on a limb. Another category of authority Hain asks the Court to look to is governmental. *See* ECF 50 at 8. Synthesizing the abundant literature, the FDA warns that lead, arsenic, and mercury harm children's neurodevelopment even at the lowest levels of chronic exposure.[17] The U.S.

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[16] *Cf.* 29 Wright & Miller § 6269.3 ("Fingerprint analysis was one of the first areas of forensics to gain widespread acceptance. As a consequence, the courts typically assume that expert testimony regarding fingerprints meets the reliability standards of Daubert and Rule 702.").

[17] *See* Brenna M. Flannery et al., *U.S. Food and Drug Administration's interim reference levels for dietary lead exposure in children and women of childbearing age*, 110 REGUL. TOXICOLOGY & PHARMACOLOGY art. 104516 at 2 (2020) ("Neurodevelopment was excluded as an endpoint in the literature review because no safe level [of lead] with respect to this endpoint has been identified to date."); U.S. FDA, ARSENIC IN RICE AND RICE PRODUCTS RISK ASSESSMENT REPORT 28 (2016) ("Children are particularly susceptible to neurotoxic effects as a result of even low-level exposure to lead and methyl mercury."), https://perma.cc/7TWA-M9Q6; U.S. FDA, *Supporting Document for Action Level for*

Department of Health and Human Services agrees.[18] Meanwhile, the Texas Department of Health and Human Services recognizes "[e]xposure to . . . lead or mercury" is one of the "[c]ommon causes" of "Non-traumatic Brain Injury," which can impair cognitive, emotional, and physical functioning. Tex. Dep't Health & Human Servs., ACQUIRED BRAIN INJURY, https://perma.cc/P7AY-7DNC (accessed Sept. 9, 2022).

Indeed, the causal connection between heavy metals toxicity and intellectual disability is so well established that it is, literally, *textbook*. *See* Bryan H. King et al., *supra*, TEXTBOOK OF PSYCHIATRY 3500 (listing "Toxins" as one of the "Postnatal Causes" of intellectual disability, and "Lead poisoning" as an example). This universal acknowledgment should not be surprising, as lead, arsenic, and mercury are three of only five chemicals "*proven* to cause developmental neurotoxicity in humans." Philippe Grandjean & Philip J. Landrigan, *Developmental neurotoxicity of industrial chemicals*, 368 LANCET 2167, 2175 (2006) (emphasis added). Thus, as Dr. Settles explained, the fact "that exposure to heavy metals in young children can have neurotoxic effects that are often permanent" is "standard knowledge most psychologists would know coming out of school." Hain Ex. 40, Settles Dep. 104:8–105:4.

Inorganic Arsenic in Rice Cereals for Infants (Aug. 2020), (FDA stating that it was unable to "quantify" a neurodevelopmentally safe level of arsenic in rice cereals for infants), https://perma.cc/6AG5-GAJG.

[18] U.S. DEP'T HEATH & HUMAN SERVS., AGENCY TOXIC SUBSTANCES & DISEASE REGISTRY, TOXICOLOGICAL PROFILE FOR LEAD 133, 136, 172-73 (2020), https://perma.cc/65RD-6AGD (observing that that even at low levels of chronic exposure, lead exposure is connected with decreased cognitive function, conduct disorders, altered fine motor skills, autistic behaviors, etc.); *id.* at 338 (advising arsenic exposure has "[g]reater-than-additive . . . neurological effects" when combined with lead exposure, "indicating a synergistic effect with" lead in harming the brain).

B. Hain's attempts to minimize the dangers of its products fall flat.

Attempting to minimize the dangers of its tainted products, Hain , Hain repeatedly plays word games in discussing the causal connection between heavy metals and autism. Hain's games only serve to highlight the ease of establishing general causation here and Hain's dim view of the vulnerable population who consume its products.

In discussing autism, Hain regularly frames the suspected causative question here as whether "baby food" is an established cause. *See, e.g.*, ECF 50 at 6, 15, 22. No case law supports Hain's level of granularity. If Hain's baby food were tainted with glass shards, an inquiry into the cause of a child's throat injury would correctly focus on the glass—the suspected immediate causative agent—not its conveyor. The same focus is required for baby food indisputably tainted with neurotoxic heavy metals, as Hain rightly does not dispute that ingestion is a well-established pathway for heavy metals toxicity.

Similarly, given that chronic exposure to even the lowest levels of lead, arsenic, and mercury exposure begin damaging a child's brain, Hain's pejorative use of "trace" to describe the level of poisons in its products, *e.g.*, ECF 50 at 1-3, reveals only its cynical disregard for children's safety. The developing brains of the young children consuming Hain's products are the most vulnerable to neurotoxins like lead, arsenic, and mercury. *See supra*, nn.17–18. At most, then, the threshold "trace" is merely the difference between chronic and acute poisoning.

Likewise, the FDA's face-saving attempt to quell panic in the wake of Congress's distressing findings about the dangers of Hain's products by surmising that, in general, "children are not at an *immediate* health risk from exposure to toxic elements in foods,"

ECF 50 at 8 (emphasis added, quotation marks omitted), is, at best, a statement about acute, not chronic risk, *see* Hain Ex. 40, Settles Dep. 148:18–149:9 (Dr. Settles explaining this distinction to Hain's counsel). All authorities, including the FDA, agree that chronic exposure to heavy metals is, in fact, quite risky to young children.

III. No Bradford-Hill (or Similar) Analysis Is Required.

Hain criticizes Plaintiffs' experts for purportedly failing to cite sufficient literature or conduct a causal review of studies sufficient to link heavy metals exposure to *autism*. ECF 50 at 30–41. But that is irrelevant, for Plaintiffs need not establish any such link. *See supra*, Part I. For good measure, however, and even though Plaintiffs' experts rely on voluminous literature in linking heavy metals toxicity to brain damage and resulting intellectual disability, Plaintiffs show that a Bradford-Hill (or similar) analysis is not a prerequisite for admissibility, let alone when general causation is beyond dispute.

A Bradford-Hill or other similar analysis of published studies is *never* required for reliability. As several courts have held, "in the context of a general causation challenge, failure to satisfy the Bradford Hill criteria does not doom admission under *Daubert*." *In re Neurontin Mktg., Sales Pracs.& Prod. Liab. Litig.*, 612 F. Supp. 2d 116, 133 (D. Mass. 2009).[19] Removing any doubt, the Supreme Court has rejected "the premise that statistical

[19] *Accord, e.g.*, *Yarbrough v. Hunt S. Grp., LLC*, 2019 WL 4392519, at *4 (S.D. Miss. Sept. 12, 2019); *Hoover v. Bayer Healthcare Pharms. Inc.*, 2016 WL 9049275, at *3 (W.D. Mo. Dec. 9, 2016); *In re Celexa and Lexapro Prods. Liab. Litig.*, 927 F. Supp. 2d 758, 766 (E.D. Mo. 2013); *Wagoner v. Exxon Mobil Corp.*, 813 F. Supp. 2d 771, 804 (E.D. La. 2011); *In re Viagra Prods. Liab. Litig.*, 658 F.Supp.2d 936, 946 (D. Minn. 2009); *see also SEC v. Life Partners Holdings, Inc.*, 854 F.3d 765, 776 (5th Cir. 2017) ("[T]he lack of scientific consensus or peer review does not necessarily render expert testimony

significance is the only reliable indication of causation," as "medical researchers consider multiple factors in assessing causation." *Matrixx Initiatives, Inc. v. Siracusano*, 563 U.S. 27, 40 (2011). Thus, "[a] lack of statistically significant data does not mean that medical experts have no reliable basis for inferring a causal link between" a suspected causal agent "and adverse events." *Id.* As a result, "medical professionals and researchers do not limit the data they consider to the results of randomized clinical trials or to statistically significant evidence." *Id.* at 41.[20]

At all events, even if a Bradford-Hill analysis were ever required to establish the reliability of a general causation opinion, it is not required in a case like this one where general causation is widely accepted (and undisputed). "The Bradford-Hill criteria 'were developed as a mean[s] of interpreting an established association based on a body of epidemiologic research for the purpose of trying to judge whether the observed association reflects a causal relation between an exposure and disease.'" *Soldo v. Sandoz Pharms. Corp.*, 244 F. Supp. 2d 434, 514 (W.D. Pa. 2003) (quoting report of court-appointed expert) (emphasis omitted). Here, there is no question of a causal relation between heavy metals exposure and heavy metals toxicity or between heavy metals toxicity and brain injury resulting in intellectual disability.

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unreliable."); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 772 (Tex. 2007) (observing that "epidemiological studies . . . are not necessary to prove causation").

[20] Although *Matrixx* was a securities case, the Supreme Court's discussion of causation is directly applicable here. *Matrixx* turned on whether undisclosed adverse events in patients taking a drug the defendant manufactured were material. The defendant argued that only a statistically significant finding of an association would be material. Several prominent amici statisticians and medical researchers, and eventually the Supreme Court, disagreed for the very reason that such evidence is unnecessary to reliably establish a causal link.

In arguing to the contrary about heavy metals and autism, Hain misguidedly relies on *Cano*.[21] The portion of *Cano* relied on by Hain, however, applied the Texas Supreme Court's opinion in *Merrell Dow Pharmaceuticals, Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997), which "was not concerned with the *admissibility* of expert testimony, but the *legal sufficiency* of that evidence," *Cano*, 362 F. Supp. 2d at 821. Unlike Hain, however, "[t]he court must . . . not conflate 'admissible' with 'sufficient.'" *Huss*, 571 F.3d at 460.

Moreover, as *Cano* recognizes, *Havner* and related cases—even though relying on the type of evidence traditionally used to show general causation—are ultimately concerned with "specific causation." 362 F. Supp. 2d at 824; *see Bostic v. Georgia-Pac. Corp.*, 439 S.W.3d 332, 351 (Tex. 2014) ("In *Havner*, we held that . . . specific causation may be established through an alternative two-step process whereby the plaintiff establishes general causation through reliable studies, and then demonstrates that his circumstances are similar to the subjects of the studies."). So *Havner*'s discussion is irrelevant to Hain's challenge to general causation testimony for that reason also.

Still more, Plaintiffs here are not resorting to *Havner*'s "alternative method of establishing causation[.]" *Bostic*, 439 S.W.3d at 347–48. That is because Plaintiffs (and their experts) have the "direct, scientifically reliable proof of causation" absent in *Havner*, 953 S.W.2d at 715. In the Texas Supreme Court's most recent decision evaluating causation in a personal injury case, it explained that direct proof of causation may consist

[21] *In re Lipitor* (cited at ECF 50 at 40), meanwhile, did not require a Bradford-Hill analysis; rather, the court merely evaluated a Bradford-Hill analysis an expert had chosen to perform. *See In re Lipitor Prod. Liab. Litig.*, 892 F.3d 624, 642 (4th Cir. 2018) (applying *Matrixx*).

of an expert's opinion derived from direct clinical experience with the plaintiff's condition, diagnostic aids, the plaintiff's medical records, and an in-person examination. *Bustamante v. Ponte*, 529 S.W.3d 447, 470 (Tex. 2017); *accord id.* at 460 ("[T]he point of *Havner* is that, even in the absence of *direct clinical experience*, a plaintiff may still establish causation through an appropriately strong associational finding.") (emphasis added).[22] As shown in the next brief, Plaintiffs and their experts have direct proof in spades.

CONCLUSION

Hain's global errors render their briefing unpersuasive and largely irrelevant. In Plaintiffs' next brief, they will show two additional global errors by Hain and demonstrate that Rule 702 allows each of Plaintiffs' challenged witnesses to offer testimony to help establish the cause of Ethan's condition.

[22] *Bustamante* also confirmed that *Havner* is a specific causation case, notwithstanding its review of evidence typically used for general causation: "Most importantly, in *Havner*, the plaintiffs could not point to facts showing 'specific causation' and were forced to rely to a considerable extent on epidemiological studies for proof of 'general causation.'" 529 S.W.3d at 469; *see also id.* at 469–70 (using "'general causation'" as a term of art in reference to a Fifth Circuit decision that does not mention general or specific causation).

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CERTIFICATE OF SERVICE

I certify that on September 22, 2022, a copy of this document was served on all counsel of record using the Court's e-filing system.

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